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TECHNICAL REPORT

SYMPOSIUM ON FORENSIC TOXICOLOGY

by

J. B. BATEMAN

July 1972

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London W.1. ENGLAND

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1. THE SYMPOSIUM

The Symposium on Forensic Toxicology was held at the Chemical defence Establishment , Porton Down , Salisbury , Wiltshire , England , on Thursday and Friday , 29 and 30 June, 1972. I am indebted to Dr. B.Ballantyne of the Pathology Section, Medical Division, CDE , for the invitation to attend. I was present only on the second day and can only offer my own version of the papers on that part of the programme, omitting Professor Camp's remarks on the future of forensic toxicology and Dr Beswick's summing-up. Details of the symposium are reproduced in the Annex. There were no precirculated abstracts. The Proceedings are to be published very promptly, probably as early as September of this year.

1.1. Paraquat Poisoning

Dr K. Fletcher gave an interesting account of the fatalities that have resulted from drinking these unusual substances, either accidentally from beer bottles placed in the fridge or deliberately with suicidal intent. Even more interesting are the factors responsible for their unique properties as herbicides and for their remarkable toxicity, still imperfectly understood.

The value of paraquat and other members of the series such as diquat and morfamquat as herbicides resides in the speed with which they are made non-toxic by the soil, so that seed can be planted immediately after spraying of weeds. This is attributed to the entry of these flat bipyridyl molecules, e.g.

$$CH_3 - {}^{+}Pyr - Pyr^{+} - CH_3 \quad .... \quad 2\ Cl^{-}$$

into the clay lattice and their extremely strong attachment by ionic and van der Waals forces. They become biologically inert and are said to be inaccessible to chemical agents and bacteria - to such an extent that they can be unveiled only by destroying the lattice with concentrated sulfuric acid. Perhaps it ought not to be surprising, therefore, that their toxicity for man is such as to suggest prolonged persistence in the body in some cryptic state. The initial action after ingestion is irritation, with gastrointestinal and renal symptoms and no apparent action on the central nervous system. However, from one to three weeks after these unpleasant effects have subsided, signs of progressive lung damage appear. Gas exchange becomes impeded, leading to cyanosis and death in about a month. The lungs have become edematous; there is thickening of the alveolar walls, intense leucocytic infiltration, fulminating fibrosis and eventually complete alveolar occlusion. The pictureiin its less extreme forms resembles the effects of oxygen at high partial pressures; indeed Dr Fletcher said privately that he has done experiments along these lines, finding that oxygen does exacerbate the toxicity of paraquat and that paraquat, like oxygen, retards wound healing. On the other hand, there is no apgparent effect on the lung lipids, although this was challenged by a person on the basis of the "bubble" test for surfactant - surely not to be taken too seriously. But why the pulmonary pathology ? There seems to be little information beyond the fact that paraquat binds to mucopolysaccharides and nucleic acids, though rather loosely. Its persistence in the body is shown by the urinary excretion, which declines rapidly for the first two days followed by a "tail" lasting for 20 to 30 days. The tissues at postmortem contain about one to five parts permillion. The LD50 for man may be around 30 mg/kg or 1 to 2 grams. For the hen it is ten times greater per kg. - why ? The remaining evidence is of a negative kind. Paraquat can be reduced to a free superoxide radical with NADPH (half life about a microsecond) but hydrogen peroxide is not likely to be involved in the toxic action. Paraquat is not immunosuppressive; carcinogenic; teratogenic; mutagenic. One would imagine that the two best leads for further work would be first to look for an analogy between the binding in the clay lattice and to biological components; second, the reciprocal potentiation of paraquat and oxygen toxicity. This might be of interest to contractors working on wound healing.

1.2. Hallucinogens

Dr. R.W. Brimblecombe classified the hallucinogens and gave a brief rundown of their characteristics, defining them as substances which specifically alter mood and thought patterns rather than acting on the autonomic nervous system or other identifiable elements. The distinction is not always easy to sustain because the hallucinogens often do exhibit other modes of activity.

The sympathomimetic drugs can either block of facilitate synaptic transmission in the central nervous system. They may produce elevated levels of 5-hydroxy tryptamine and depression of brain noradrenalin. There may be marked cross tolerance among members of the group. Two structural subgroups exist; the N,N-dialkyl tryptamines, including psilocin and LSD, and the phenyl ethyl amines with mescaline and some substituted amphetamines such as the celebrated "Security, Tranquillity and Peace," STP.

The anticholinesterases, atropine, hyoscine, piperidyl benzoates, etc., act centrally by competitive antagonism of acetylcholine-esterase and by other mechanisms. Unlike LSD they produce a true toxic psychosis with amnesia.

The cannabinoids, with the 8-delta and 9-delta tetrahydrocannabinols as the main active principles, produce central depression with the cerebral cortices as probable sites of action - but little is known.

1.3. Diagnosis of Acute Cyanide Poisoning

Dr. B. Ballantyne pointed out that since cyanide poisoning cannot well be studied in man, diagnosis depends largely upon what has been learned from animals - and, he might have added, from classical studies of respiration in much more primitive systems. Cyanide acts not only as an inhibitor of cytochrome oxidase. It also inhibits glutamic decarboxylase and so produces convulsions. Respiratory depression prevents utilization of oxygen so that the blood remains red - a characteristic post mortem sign. Chemical tests for cyanide may mislead because of post mortem formation of cyanide from thiocyanate even in sterile blood. Cytochrome oxidase activity in tissue sections can be determined spectrophotometrically by formation of a dye with a special reagent.

1.4. Mercury Poisoning

Dr. P.L. Bidstrup dealt with the comparative aspects of the toxicity of mercury and its compounds, drawing a distinction between metallic mercury, inorganic salts, and the alkyl and aryl compounds.

Metallic and inorganic mercury are used in some 80 industries in about 3000 different ways. The key fact that a patient works with mercury may be overlooked, sometimes with bizarre results, as Dr Bidstrup showed by a series of anecdotes. One was about an electric meter repair man whose irritability, withdrawal and tremor were first considered psychotic and then attributed to multiple sclerosis. The patient eventually diagnosed his own complaint after reading in the newspaper about mercury poisoning. The hazards from metallic mercury are still insufficiently recognized by teachers and others although it is obvious that small amounts spilled may remain in a finely divided state, with relatively enormous surface area for evaporation, in cracks and corners of the room or in rugs or carpets where children may crawl. The danger is greater in warm places, the vapour pressure having a temperature coefficient of 2 for 10°C. Further, mercury vapour being soluble in fat, it reaches the brain. Inorganic salts, on the other hand, produce kidney damage, although the nephrotic syndrome is now rather rare. The usual signs nowadays are erethism and tremor, the latter leading to solitary habits because of fear that clumsiness caused by tremor will be noticed. Pneumonitis is the usual cause of death. Various special forms may be encountered - e.g., allergy to cinnabar and hypersensitivity causing "pink disease" in children.

The aryl mercury compounds mainly cause skin symptoms. They were not discussed in any detail, although their presence in food is a cause of much current poisoning.

The alkyl mercury compounds are extremely dangerous, producing cerebral cortical atrophy revealed at an early stage in narrowing of the visual field through atrophy of the area striata. There is no motor nerve involvement but there is degeneration of peripheral sensory nerves. According to Swedish investigators genetic effects are suspected because chromosome breaks have been found in people who have eaten fish containing high mercury levels. The lowest intake rate likely to produce symptoms has been estimated to be 0.3 mg Hg/70 kg.x Day leading to a concentration in the blood of about 0.2 microg./g. This is an improbable but not impossible rate of intake for people who eat a lot of fish. As for fatal cases, the most have been in Japan where the food chain resulted in high mercury levels in fish in an area of industrial pollution, and in Iraq where mercury treated grain intended for planting was instead baked and eaten. During the discussion of this paper there was caustic comment on the folly of sending such grain to underdeveloped countries where its diversion from the farm to the bakery would be very likely to happen.

1.5. A Field Kit for Drug Detection

Professor E.G.C.Clarke gave a well staged demonstration of his box of tricks for the rapid determination of drugs in urine, prefacing the show by the remark that mass spectrometers and skilled operators are not always available when and where they are most needed. The box contains, essentially, a few small bottles, a number of reagents, strips of filter paper and small tiles. The original procedure, published in the British Medical Journal, has now been extended. I do not recall all the steps but the following will give some idea. The urine sample is extracted with chloroform in presence of solid sodium sulfate and the extract spotted in several places on a strip of filter paper. Iodoplatinate gives a colour test for morphine and nicotine, distinguished from methadone and some others by adding sodium sulfite and noting the colour change. Paraldehyde in concentrated sulfuric acid is used for morphine and other substances and mixtures are resolved by ten minute paper chromatography. The benzodiazepin drugs - librium, etc., -are done either by diazotization and coupling or (in the case of librium) by lactam formation followed by hydrolysis to give a common end product. The full description will be given in the published proceedings, possibly amplified by inclusion of the barbiturates.

1.6. Anticholinesterase Poisoning

It was unfortunate that the only paper of the day that placed any significant mental demands on the audience was that of Dr J.M.Barnes, read in his absence by Dr Beswick immediately after the luncheon break, when the effects of an early start from London began to make themselves felt. Dr Barnes began by saying (by proxy) that the field of the anticholinesterases is full of riddles and that it is of little use to cling to oversimplifications and preconceptions when some honest intellectual effort is what we need. All to no avail; the only thing that can be reported about this excellent paper is that it stressed the delicate balance between acetylcholine and cholinesterase levels, regulated both in terms of rates of change as well as steady state degrees of inhibition, and the importance of adaptative reactions which can lead to more or less normal functioning even when blood AcCh levels are permanently elevated, on the one hand, or when blood esterase is zero, on the other, as may occur in myasthenia gravis (presumably during anticholinesterase therapy). In the ensuing discussion it was questioned whether acetylcholinesterase is really essential to the functioning of the central nervous system since life can continue

in its absence while antiesterases can lead to death even when the esterase level remains high. There was some discussion of the specific, non-specific and pseudo cholinesterases, the last of which are not true esterases.

1.7. Organophosphate Residues In Soil and Vegetation

In this paper Dr. T.D. Inch dealt mainly with analytic procedures without going into their application in the study of the mobility of insecticides in soil and the persistance of breakdown products in vegetation, except to say that they are in fact extremely long-lived. He added that experiments on uptake from the vapour phase encourage the belief that plants may prove useful in pollution assay.

The methods in use are mainly gas chromatography with or without mass spectrometry. The thermionic detector for phosphorus is extremely sensitive (one part P in 10^9 parts soil- far less sensitive in vegetable matter) but there is a need for other selective detectors. Thin layer chromatography has been developed in a very clever way using TLC plates formed from films of finely divided soil. The bands are assayed either using labelled compounds or by eluting slices for gas chromatography.

1.8. Proof of Teratogenicity

In his excellent paper, Dr. Sullivan was at pains to show that in the slippery field of teratogenics there are few conclusive proofs; for every bit of seemingly watertight argument an exception can be found in practice.

There are two aspects of the problem: one is the testing of a drug for possible teratogenic effects; the other, determining the cause of an actual malformation. Dr. Sullivan dealt mostly with situations in which both aspects are presented: the occurrence of malformations for which one or more candidate drugs enter the picture.

Three questions must be asked:

a. Was the patient exposed to the suspected drug?
b. Did the exposure occur at the appropriate time?
c. Did the suspected agent actually produce the observed effect?

a. Proof of exposure: This can be considered first by looking at epidemiological data, and then by means of personal interviews.

Both have their pitfalls. In the first epidemic in Hamburg, the incidence of malformations between 1957 and 1962 correlated beautifully with the sales volume of the drug thalidomide, and a similar relationship was later found for the rest of Germany. However, the number of mothers with malformed babies who denied having taken the drug likewise was greatest when sales and malformations were most frequent. The shortcomings of the interviewing technique were apparent again during the epidemic in Sterlingshire when not only the mothers but also their physicians denied that thalidomide had been used. Recourse to the National Health Service and inspection of prescriptions filled showed that these statements, with one exception, were untruthful.

b. Exposure at the right time? The effects of toxic agents during pregnancy are critically dependent upon timing. In the mouse it has been shown that exposure to anoxia for one day, for example, produces intrauterine death with major structural abnormalities if it occurs during the period of embryogenesis following implantation. If imposed during the later stages of foetogenesis, intrauterine death still occurs but the abnorm alities are functional, not structural. Thus the suspect drug can only be found guilty if the nature of the abnormalities agrees with what would be predicted from the dates upon which it was taken. Although this is a good rule, it is not infallible. Secondary rupture of a fused palate can occur and neuropore closure might also be reversed. Again, some defects become apparent only after birth. This is true of heart defects and of the association between the much later development of leukemia and infection of the mother with influenza during pregnancy. It is especially important where carcinogens are concerned. Cancers of the central nervous system produced in rats by the nitrosamines, for example, show increasing frequency with increasing age, linear on a double log plot. Obviously it becomes very difficult in human populations to prove the association between an agent encountered prenatally and a disease which only appears later in life.

c. Direct proof of drug effect: This is often extremely difficult. Dr Sullivan gave examples of apparent teratogenicity in which it was unlikely that the drug would be able to cross the placenta, so that any effect on the foetus would have to be secondary. For example, serotonin and trypan blue probably act either by reducing uterine blood flow or by inhibiting the trophoblast enzymes. A striking demonstration of direct action, on the other hand, was shown in the teeth of a child whose mother had been treated several times during pregnancy with tetracycline. These treatments could be detected in tooth sections in the form of rings of decreased deposition. Sullivan also cautioned against false accusation of harmless drugs. It had been asserted that progesterone given to prevent premature termination of pregnancy led to anencephaly, spina bifida and other abnormalities; but experimental studies led to the conclusion that these were the consequences, not of excessive dosage of progesterone, but of insufficient.

d. Criteria for direct testing: Sullivan has computed the specifications of a program for detecting teratogenicity in humans. If the normal incidence of malformations is one per thousand, he calculates that 23,000 women would have to take part in the experiment in order to detect with acceptable reliability a teratogenic effect sufficient to double the frequency of defects. Even to identify active drugs with only 50% reliability the number of "subjects" required would be about 8,000. Clearly there is little hope of being able to do tests on such a scale, and no solution to the problem seems to be in sight. Mention was made of an NIH project for recording all birth defects and other items considered relevant in a large population, but the computer evidently has its own birth defects, having failed to notice an epidemic of rubella of which all local physicians and most of the pregnant women were well aware. Dr Sullivan's conclusion was that there is still no substitute for the alert individual observer.

2. SUMMARY

A symposium on Forensic Toxicology was held at the Chemical Defence Establishment, Porton Down, on 29 - 30 June, 1972. In this report eight of the verbal presentations given in the second half of the program are summarized, dealing with (a) paraquat poisoning, (b) hallucinogens, (c) cyanide poisoning, (d) inorganic and organic mercury toxicity, (e) anticholinesterases, (f) organophosphate residues, (g) teratogenicity. Special mention is made of (g), Professor E.G.C.Clarke's field kit for drug detection which is remarkably sensitive and selective and should be of interest in the U.S. drug research program.

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Dr A C MOFFAT
Senior Research Fellow, Home Office Central Research Establishment, Aldermaston

THURSDAY 29TH JUNE

MORNING SESSION

CHAIRMAN - Professor F E CAMPS

0930	Introductory talk by Mr G N GADSBY, CB, Director of the Chemical Defence Establishment	
0940	Dr A S CURRY	Methodology and Interpretation in Forensic Toxicology
1010	Dr A C MOFFAT	The Use of Enzymes in the Detection of Drugs
1040	Dr ANN E ROBINSON	Postmortem Morphine and Methadone Levels
1110	BREAK	
1130	Dr A D BEATTIE	Clinical and Biochemical Aspects of Lead Poisoning
1200	Dr M H P SAYERS	Screening for Lead Poisoning
1230	LUNCH	

AFTERNOON SESSION

CHAIRMAN - Dr A S CURRY

1400	Mr J M JACKSON	The Poisoned Meal
1430	Dr R GOULDING and Mr J GROVE	Non-fatal Poisoning: the Hospital and the Laboratory
1500	BREAK	
1530	Dr D J GEE	Barbiturate Poisoning
1600	Flt Lt D J BLACKMORE	Interpretation of Carbon Monoxide Levels Found at Postmortem
1630	GENERAL DISCUSSION	

CHAIRMAN - Dr J M BARNES

0930	Dr K FLETCHER	Paraquat Poisoning
1000	Dr R W BRIMBLECOMBE	Hallucinogens
1030	Dr B BALLANTYNE	The Diagnosis of Acute Cyanide Poisoning
1100	BREAK	
1130	Dr P LESLEY BIDSTRUP	The Comparative Toxicity of Mercury Compounds
1200	Professor E G C CLARKE	Rapid Tests for Drugs in Urine
1230	LUNCH	

AFTERNOON SESSION

CHAIRMAN - Dr F W BESWICK

1400	Dr J M BARNES	Anticholinesterase Poisoning
1430	Dr T D INCH	Organophosphorus Anticholinesterase Residues in Soil and Vegetation
1500	BREAK	
1515	Dr F M SULLIVAN	Proof of Teratogenicity
1545	Professor F E CAMPS	The Future of Forensic Toxicology
1615	Dr F W BESWICK	Concluding Remarks

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DOCUMENT CONTROL DATA - R & D

(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY (Corporate author) US Army Research and Development Group (Europe), Box 15, FPO New York 09510	2a. REPORT SECURITY CLASSIFICATION Unclassified 2b. GROUP

3. REPORT TITLE

Symposium on Forensic Toxicology

4. DESCRIPTIVE NOTES (Type of report and inclusive dates)

Technical Report, July 1972

5. AUTHOR(S) (First name, middle initial, last name)

John B. BATEMAN

6. REPORT DATE July 1972	7a. TOTAL NO. OF PAGES 15	7b. NO. OF REFS 0
8a. CONTRACT OR GRANT NO. na b. PROJECT NO. na c. d.	9a. ORIGINATOR'S REPORT NUMBER(S) ERO - 10 - 72 9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report) 1972 t	

10. DISTRIBUTION STATEMENT

Approved for public release; distribution unlimited.

11. SUPPLEMENTARY NOTES	12. SPONSORING MILITARY ACTIVITY US Army R and D Group (europe) Box 15, FPO 09510 New York

13. ABSTRACT

A symposium on Forensic Toxicology was held at the Chemical Research Establishment, Porton Down, UK, on 29 - 30 June 1972. In this report eight of the verbal presentations given in the second half of the program are incompletely summarized, dealing with (a) paraquat poisoning, (b) hallucinogens, (c) cyanide poisoning, (d) inorganic and organic mercury toxicity, (e) anticholinesterases, (f) organophosphate residues, (g) teratogenicity. Special mention is made of Professor E.G.C.Clarke's field kit for drug detection which is remarkable sensitive and selective.

Key words: toxicology, forensic; paraquat; hallucinogens; mercury poisoning; cyanide poisoning; anticholinesterases; teratogenicity, proof of; drug detection, field kit.